Socio-demographics, Clinical profile and Disposition of In-patients with Acute Decompensated Heart Failure at Kenyatta National Hospital

A dissertation submitted as part of fulfilment for the degree of Master of Medicine in Internal Medicine

By

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DECLARATION

This dissertation is my own original work and has not been presented for a degree in any other university.

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This dissertation has been presented with our approval as university supervisors.

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DEDICATION

This work is dedicated to my late father Zachariah Sikhira who died of heart failure and to all the people living with heart failure throughout the world.

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I would like to thank the following for their contribution towards the success of this work:

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ADHERE...Acute Decompensated Heart Failure Registry

ADHF……Acute decompensated heart failure

AHA……. American Heart Association

ACC……..American College of Cardiology

ACE……...Angiotensin Converting Enzyme

Hb……..Haemoglobin

CCF……..Congestive Cardiac Failure

CI……..Confidence interval

CTR……..Cardiothoracic ratio

Echo……..Echocardiogram

HF…….Heart Failure

KNH……..Kenyatta National Hospital

LV…….Left ventricle

MI …….Myocardial Infarction

Min…….Minute

mmHg…… millimetres of mercury

NYHA……New York Heart Association

RAAS ……Renin –Angiotensin-aldosteron system

SD.....Standard deviation

PI……Principal Investigator

USA…….United States of America.

WHO……..World Health Organization

1. ABSTRACT

Background

Heart failure is a major clinical and public health problem worldwide due its high morbidity and mortality. It is a disease of the elderly in the developed countries. Valuable information concerning its epidemiology, clinical and laboratory characteristics is scanty in Africa in general and Kenya in particular. We conducted a study to determine the prevalence and evaluate the socio-demographics, clinical characteristics and disposition of in-patients with acute decompensated heart failure at Kenyatta National Hospital - a tertiary medical centre in Nairobi, Kenya.

Objective

To determine epidemiologic features, clinical profile and non- pharmacological level of care in heart failure patients admitted at Kenyatta National Hospital.

Design

Prospective Clinical observational register of patients admitted with acute decompensated heart failure over a period of six months.

Setting

Medical wards at Kenyatta National Hospital, Nairobi, Kenya.

Subjects

Two hundred and eighty six patients aged thirteen years and above admitted with acute decompensated heart failure. Diagnosis was based on the Modified Framingham Clinical Criteria for the Diagnosis of Heart Failure.

Results

A total of 5,043 patients were admitted into the medical wards over the six months period (December 2007- may 2008). Of these, 458 patients were screened and 286 recruited giving a prevalence of 5.7%. Female comprised 53.8% and males while 46.2%. The mean age was 44 years. Their districts of birth and residence over the past 5 years were mainly Nairobi and Central Kenya (province) districts. Two hundred and five (71.6%) had attained none or only primary education. One hundred and four (54%) were married (54%) and 111 (39.9%) were employed. Eighty five of the (29.7%) had history of alcohol consumption while 66 (23.2%) had history of tobacco smoking. Two hundred and sixty nine patients (94.2%) were admitted in NYHA functional classes III and IV. The mean haemoglobin level was 12.92g/dl while their median creatinine was 97micromol/l. The mean serum sodium concentration was 137 mmol/l, with 102 (35.8%) having Hyponatriema. The mean serum potassium concentration was 4.8mmol/l. The commonest radiological findings were cardiomegally (97.3%) and alveolar oedema (81.4%). Non pharmacological treatment was poorly offered. In-hospital mortality was 10.8%.

Conclusion

The six month period prevalence of acute decompensated heart failure in medical in-patients at KNH was high at 5.7%. Both sexes were affected equally. It is a disease of all age groups with a peak in the 20-40 year age bracket. Majority of patients were admitted with NYHA class III and IV symptoms and were fluid overloaded. Non pharmacological modes of therapy offered at discharge were significantly sub-optimal.

2. INTRODUCTION AND LITERATURE REVIEW

2.1 Background

Heart failure is defined as a state in which there is impaired ability of the heart to fill with or eject blood at a rate commensurate with the needs of the body's metabolizing tissues (1). The heart has a big reserve and coupled with various compensatory mechanisms, cardiac function is well maintained and patients remain asymptomatic till the late stages of the disease.

Heart failure is a major clinical and public health problem and like other non-communicable diseases like diabetes mellitus and hypertension, its incidence is increasing globally. Indeed, the World Health Organization (W.H.O.) estimates the global annual incidence of HF at 15 million cases (2). Improved control of infectious diseases, increased life expectancy and rapid socio-economic changes in the developing countries are some of the factors contributing to the rising prevalence of this condition. More recently, the prevalence of ischemic heart disease/failure patients has risen due to advanced management protocols for myocardial infarction that have significantly reduced mortality in this group and culminated into more survivors (3). In Kenya *Oyoo et al* in (1993) found out that the prevalence of heart failure stood at 3.3% of all medical admissions (at the national referral hospital) (4). Europe, with a total population of 600 million, has 10 million people living with heart failure and the cost of their care amounts to 20 billion Euros directly (indirect costs are much more). Besides, the rate of hospitalization for heart failure in the continent rose from 50 in 1970, to 250 per 100,000 population in1996 (5). In the United States almost five million patients are living with heart failure and nearly a half a million new cases are diagnosed with the condition for the first time every year. The annual budget for treatment of heart failure patients in the country (USA) is about 29 billion US dollars (6).

Heart failure is associated with an unacceptably high mortality; only comparable to malignant disease. 25- 35% of patients with heart failure have been shown to die annually in community surveys while hospital mortality ranges from 2 - 3% in peripheral health units to 19% in referral hospitals. The five year mortality remains at 50% despite newer strategies of management of the condition (7).

Treatment of heart failure has evolved over the past two decades due to better understanding of its pathophysiological mechanisms. Most patients are therefore able to enjoy a good quality of life. Unfortunately, most clinicians tend to concentrate on drug therapy while ignoring simple non-costly measures such as diet modification, weight management, smoking cessation, avoidance of excessive alcohol consumption and exercise which if not addressed may make the condition refractory to treatment.

2.2 Epidemiology of heart failure

The magnitude of the problem of HF can not be assessed with precision since reliable, population based estimates of its prevalence, incidence, and prognosis are lacking. Part of the problem is that large differences exist among studies in their definition of the condition and the methods used to establish its presence. In addition, presymptomatic left ventricular dysfunction is now used increasingly as an indicator of impending, if not existing, HF (8,9). The Framingham Study, renowned internationally for its contribution towards the broad topic of cardiovascular medicine in a community setting, is primarily based upon symptomatic HF. The Study found prevalence in men of 8 per 1000 at age 50 to 59 years, increasing to 66 per 1000 at ages 80 to 89 years; similar values (8 and 79 per 1000 respectively) were noted in women. Its findings and figures do not therefore include asymptomatic patients with a reduced LVEF (10). Investigations using echocardiography have found that only 50% of participants with left ventricular dysfunction are symptomatic. In a community survey from the Mayo Clinic, for instance, only 24% of patients with LVEF of less than 50% had a clinical diagnosis of HF (11).

There has been an increase in the prevalence of HF in the population over time. In the REACH (Resource Utilization Among Congestive Heart Failure) study conducted over a ten year period (1989- 1999) in Kansas USA, the average annual increase was 1/1000 and 0.9/1000 for women and men, respectively. This finding was associated with a fourfold rise in the rate of hospitalization for HF over the study period. Some of the factors contributing to this scenario were thought to be improved treatment of hypertension, valvular and coronary diseases thus allowing patients to survive an early death only to later develop HF (12). However, these trends must be interpreted with caution because of the introduction of new diagnostic methodology, changes in hospital admission practices, increased awareness of the problem; and changes in the prevalence of co-morbidities. Coexistent disease is often the chief reason for hospitalization of HF patients.

Epidemiological studies elsewhere across the globe show interesting disparities in terms of the various socio-demographic aspects of heart failure. In Kenya, a prospective study by *Ogola* et al conducted in 1991 found out that 39.5% of patients aged 60 years and above admitted at Kenyatta National Hospital had echocardiographic evidence of cardiovascular disease/ heart failure (13). In a European study (2000), R*ywick et al* reported a more than 50% prevalence of heart failure in patients above 65 years attending out patient clinics in Warsaw, Poland (14). In the Arab population heart failure as a condition is exceptionally rare: a prevalence of 5.17/1000 population was found in a University hospital (Sultan Qaboos Oman) by *Agarwal et al* in 2001 (15).

Sex and age distribution of heart failure patients across the globe also reveal great differences especially regarding the latter. Whereas HF patients in developing countries are young, the condition is primarily a disease of the elderly in the Western/Developed World. In the Kenyan Study by *Oyoo et al*, the mean age of the patients was 39 years while *Amoah et al* in Ghana (2000) reported a mean age of 42.3 years (16,17). In a more recent study in Ghana (2007) *Kofi I. Owusu* reported a mean age of 53.9 years (18). In Portugal *Ferreira et al* (1996) reported a mean age of 69 years in a retrospective analysis of HF patients in a tertiary hospital over a 6 year period (19). Figures from the Arab world share similarities with the west: prevalence of 1/1000 in persons aged less

than 45years to 25/1000 in those aged 45 - 64years in a study by *Agarwal et al* (15). Unexpectedly, in a prospective study conducted in a University Teaching Hospital in Iloria Nigeria by *Isezuo et al* in 2003, the one year mortality in the young patients (below 40 years) was significantly higher compared to the older population (20). Most studies however, quote an equal sex distribution, with women having a slightly higher margin over men except in the Arab population where the condition is predominantly a disease of men (15).

The geographical distribution of heart failure in Kenya is unknown. This information may be important in terms of resource allocation and planning of preventive strategies for HF. This study will provide insights into this particular aspect. On a continental scale however, the good news is that a multi-centre prospective study - The Thesus Protocol – will soon start in four different settings: two hospitals in S. Africa, one in Cameroon and one in Maputo, Mozambique (which will be the coordinating centre). This study, funded by the Duke Clinical Research Institute, USA, will seek to characterize the epidemiology of heart failure and its geographical variation in Africa (17).

Residence in particular areas may also play a role in the evolution and subsequent development of heart failure in some settings. Cardiomyopathies in general are endemic in Africa; and endomyocardial fibrosis (EMF) in particular, it is restricted to the wet forest regions of the East, Central and West Africa. Although the pathogenesis of EMF is not fully understood, it appears that the conditioning factors are geography and diet, the triggering factor being yet an unidentified infective agent (17).

2.3 Clinical aspects of heart failure

Heart failure as a clinical syndrome results from any structural or functional cardiac disorder that impairs the ability of the ventricles to fill with or eject blood. The cardinal clinical manifestations are dyspnoea and fatigue (that limit exercise tolerance) and fluid retention - that may lead to pulmonary and peripheral oedema. These abnormalities may not necessarily dominate the clinical picture at the same time (21). Several scoring systems based on symptoms and signs have been developed to assess the presence and severity of heart failure: NHYA, Boston, Walmas, Gheorgiades and 'Men born in 1913'. *Mostred et al* in Netherlands reviewed these systems in terms of sensitivity and specificity and concluded that they all possessed high sensitivity but lacked the capacity to detect early or pre-clinical HF (22). New York Heart Association (NYHA) classification system is the most validated system hence commonly used internationally.

Recent evidence from studies based in the general population, general practice and hospital strongly suggest that majority of men and women with left ventricular systolic dysfunction are asymptomatic (9). This finding, together with the beneficial effect of angiotensin-converting enzyme inhibition in these patients reported in randomized trials, has fuelled discussions regarding the potential of echocardiographic screening of large populations or high-risk patients. Moreover, the potential additive value of echocardiographic or neurohumoral assessments in general practice remains to be established in more clinical epidemiological studies. The current concept therefore, is one that emphasizes both the evolution and progression of the disease. This is the system adopted by the American Heart Association and the American College of Cardiology. It identifies 4 stages of heart failure (1):

Stage A: Patients who are at risk of developing heart failure but have no structural disorder of the heart.

Stage B: patients with a structural disorder of the heart but who have never developed symptoms

Stage C: Patients with past or current symptoms of heart failure with associated structural heart disease.

Stage D: patients with end stage disease that requires specialized treatment strategies.

Whereas the AHA/ACC system of classification of heart failure is more relevant in public health management, the NYHA system has traditionally been used in clinical research settings. A Polish study in an out-patient setting by *Rywick et al* found out that only a third of the patients were in the NYHA class III and IV at Diagnosis (14). A similar Kenyan study by *Oyoo et al* on in-patients reported that more than 90% of them were in classes III and IV (4).

A comprehensive study on various clinical aspects of HF by *Mclister el al* in Canada revealed interesting findings contrary to the widely held view. Majority of the patients had a normal heart rate with an average of 80 beats/min and were normotensive with a mean systolic BP of 123mmHg and diastolic BP of 75mmHg. In the same study he showed that only 5.1% of had peripheral limb oedema (23).

2.4 Radiological and selected laboratory features in Heart Failure

A chest X-ray is a useful first diagnostic test, particularly in the evaluation of patients who present with dyspnoea, to differentiate HF from primary pulmonary disease. Findings suggestive of HF include cardiomegally (cardiac-to-thoracic width ratio above 50 %), cephalization of the pulmonary vessels, Kerley B-lines, and pleural effusion. The cardiac size and silhouette may also reveal signs of congenital anomalies (ventricular or atrial septal defect) or valvular disease (mitral stenosis or aortic stenosis) (24).

A systematic review of the utility of a chest radiograph to diagnose LV dysfunction by *Badget et al* concluded that cephalization and cardiomegally were the best predictors of increased preload and reduced ejection fraction respectively (25). Neither finding, however, was sufficient to make a definitive diagnosis of HF. In a multicenter study of 880 patients by *Knudsen et al* in Norway, the combination alveolar oedema, interstitial

oedema, and cephalization had a specificity of more than 90% for HF. Isolated cardiomegally on the other hand recorded a sensitivity of 50 % (26).

Anaemia is a frequent finding in patients with heart failure, with a prevalence ranging from 4% to 55% depending upon the population studied (27). In an analysis of the SOLVD (Studies of Left Ventricular Dysfunction) database in Boston USA, for example, 26% of the patients had their haematocrit value below 39% (28). A similar rate of anaemia (17%) was noted in a population-based cohort of 12,065 patients with newly diagnosed HF (27). *Silverberg et al* in Israel, showed that the incidence of anaemia appears to increase with worsening functional class (from 9% for NYHA class I to 79 % for class IV (29).

The aetiology of anaemia in heart failure is multifactorial. Increased circulating cytokines such as tumour necrosis factor-alpha is a significant contribution via myelosuppresion just like in other chronic diseases. Haemodilution from plasma volume expansion and malnutrition in advanced HF due to chronic liver congestion (resulting to cardiac cachexia) are other known factors. Iron deficiency is also a significant cause of anaemia in HF patients. In a recent series in Greece (Athens), *Nanas et al* showed that iron deficiency contributed to up to 70% of the causes of anaemia in advanced HF (30). The exact cause of the iron deficiency is not clear. Angiotensin converting enzyme (ACE) inhibitors, which prolong survival in patients with HF, also appear to induce anaemia in selected patients. The impact of ACE inhibitors was evaluated in a report from the SOLVD trial in which patients with left ventricular dysfunction were randomly assigned to enalapril or placebo. At one year after randomization, the rate of new anaemia (hematocrit 39% in men and 36%) was significantly higher in the enalapril group (11.3 versus 7.9% with placebo with an adjusted odds ratio of 1.56). The difference in hematocrit between the two groups was evident as early as six weeks of treatment (31). The adverse effect of ACE inhibitors may be mediated by Ac-SDKP (goralatide), a tetrapeptide that inhibits erythropoiesis. Ac-SDKP is metabolized by angiotensin converting enzyme and would be expected to accumulate in the presence of an ACE inhibitor, thereby inhibiting erythropoiesis.

Correction of anaemia in HF is associated with significant benefits: lower mortality (up to 25%), improved NYHA functional class (up to 42%), increased left ventricular ejection fraction (up to 5.5%), reduced requirement for oral or intravenous diuretics and fewer days of hospitalization (29).

Renal function (serum electrolytes and creatinine) is another important baseline test before initiating therapy with diuretics and/or ACEIs in HF. Besides, HF resulting from ischemic/ coronary heart disease is a common cause of morbidity in patients with end stage renal disease (ESRD) (32).

2.5 Pathophysiology of heart failure

A healthy adult heart pumps out 5 litres of blood per minute – translating to 7,200L/ per day (assuming a stroke volume of 66ml, at 72 beats/minute at rest). In cases of increased tissue demand, there is increased venous return, increased diastolic filling, increased ventricular stretch / volume and increased contractility (Starling law of the heart). Normal physiology requires a healthy myocardium, competent valves, an intact conducting system and a normal peripheral resistance (33).

Cardiac dysfunction precipitates changes in cardiac size, vascular function, blood volume and neurohumoral status in an attempt to maintain cardiac output and arterial blood pressure (so called compensatory mechanisms). However, these factors tend to worsen cardiac function and indeed some of the most effective treatments of heart failure involve their pharmacological manipulation. Generally, as heart disease progresses into HF, there is an increment in cardiac size (called cardiac or ventricular remodelling) that is paradoxically associated with further deterioration in cardiac function before symptoms of HF become evident (33). It is manifested clinically by changes in cardiac size, shape, and function in response to cardiac injury or increased load. The importance of

remodelling as a pathogenic mechanism is incompletely understood, since the factors that cause the phenomenon are actually determinants of HF prognosis rather than ventricular dilation. Consistent with the hypothesis that remodelling is pathologically important in HF is the observation that ACEI inhibitors and beta blockers improve survival in patients by slowing and in some cases even reversing the process (34).

Other changes in cardiac function associated with heart failure result in a decrease in cardiac output due to systolic and diastolic dysfunction. Systolic dysfunction results from loss of intrinsic inotropy due to alterations in signal transduction mechanisms within the myocardium or loss of viable contracting muscles as occurs during acute myocardial infarction. This process is accompanied with a higher ventricular end diastolic pressure, which serves as a compensatory mechanism by utilizing the Frank-Sterling mechanism to augment stroke volume.

Neurohumoral responses include over stimulation of sympathetic nerves, renin - angiotensin –aldosteron system (RAAS), vasopressin, endothelin and atrial natriuretic peptide. Their net effect is to produce arterial vasoconstriction in order to maintain blood pressure (35). These changes can be viewed as compensatory mechanisms but they also aggravate heart failure by increasing both ventricular after load and preload. The RAAS (particularly angiotensin II) is directly implicated in the pathogenesis of ventricular remodelling that has deleterious effects as discussed (34). Atrial natriuretic peptide (ANP) is primarily released from the atria in response to volume expansion, which is sensed as an increase in atrial stretch. Plasma ANP levels rise early in the course of the disease and have been used as a marker for the diagnosis of asymptomatic left ventricular dysfunction. With chronic and more advanced heart failure, ventricular cells can also be recruited to secrete both ANP and brain natriuretic peptide (BNP), an analogous peptide, in response to the high ventricular filling pressures. These relationships have allowed the plasma concentration of these peptides, particularly BNP, to be used in both detection and prognostication of heart failure (36). Endothelin, another substance produced by the vascular endothelium, may contribute to the regulation of myocardial function, vascular tone, and peripheral resistance in HF. Plasma endothelin concentrations are increased in

patients with HF and angiotensin II may contribute to its high circulating levels. In the long-term however, high levels of endothelin may be deleterious to the heart due to its contribution to pathologic remodelling and increased ventricular after load. (37).

Increase in blood volume in HF serves to increase ventricular preload and thereby enhance stroke volume by the Frank-Sterling mechanism. With time, ensuing pump failure leads to reduced renal perfusion with resultant oliguria and fluid retention. With further hypoperfusion, the sympathetic nervous system and the RAAS are activated. There is also an increase in circulating arginine vasopressin contributing to renal water retention (38). The final outcome of these mechanisms is an increase in renal re-absorption of sodium and water. The resultant increase in blood volume helps to maintain cardiac output, but is deleterious because it raises pulmonary and systemic venous pressures leading to pulmonary and systemic congestion.

2.6 Diagnostic evaluation of heart failure patients

Diagnosis of acute decompensated heart failure is based on a constellation of clinical symptoms and signs in conjunction with characteristic imaging findings (electrocardiogram, chest radiograph and echocardiogram). The clinical features usually result from pulmonary and venous congestion in addition to exercise intolerance. The '***Modified Framingham Clinical criteria for diagnosis of Heart Failure'*** (modified in 1998 from the original criteria that that was published in 1972) utilizes both clinical and chest radiographic features to confirm the presence of HF and has been used as the gold standard in most published research settings (Appendix 1).

From the history two major classes of symptoms of heart failure prevail: fluid retention (characterized by oedema, orthopnea, hepatic congestion and ascites) and reduction in cardiac output (characterized fatigue, dyspnoea and weakness). Absence of dyspnoea

especially on exertion virtually rules out HF (39).The history will also provide vital clues concerning the aetiological basis of the syndrome.

A thorough physical examination will pick features of fluid overload such as oedema, ascites, pleural effusion, raised jugular venous pressure and pulmonary crackles. Examination of the heart may reveal a displaced apex, murmurs and S3, S4 or both (summation gallop). Other feature like pallor or stigmata of infective endocarditis may be seen especially if anaemia and the latter are the precipitants of acute decompensation.

A chest radiograph may reveal features of pulmonary venous congestion such as alveolar oedema and Kerly B lines; and cardiomegally. Pleural effusion is often present but may be absent in acute heart failure (40). Electrocardiography (ECG) may reveal underlying conditions that predisposed the patient to HF such as LVH, myocardial ischemia, pericarditis and atrial fibrillation (41).Echocardiography should be performed to all patients with HF. It provides important information on left ventricular size and function besides establishing the aetiology of HF. The sensitivity and specificity of the standard two-dimensional echocardiogram for diagnosis of HF is 80% and 100% respectively (42).

Laboratory data should be routinely obtained in all patients with HF. They are not strictly necessary for making a diagnosis of HF but they may be useful in guiding initial therapy. Arterial blood gases may show hypoxemia. Full blood counts may reveal anaemia or infection as precipitants of acute decompensation. Routine chemistries may reveal renal dysfunction due to low cardiac output or as a pointer to the underlying renal disease. B-type natriuetic peptide (BNP) and N- terminal BNP assay may also supplement clinical judgement when the cause of a patient's dyspnoea is uncertain (43). Finally, if there is suspicion of ongoing myocardial ischemia, cardiac enzymes can be measured to rule out myocardial injury (1).

In the differential diagnosis of patients with suspected HF, pulmonary thrombo-embolism, pneumonia and myocardial ischemia should be considered. These can usually

be ruled out by a focussed history, physical findings and appropriate laboratory investigations.

2.7 Treatment of heart failure

Diet, exercise, cessation of smoking, avoidance of excessive alcohol consumption and reduction of both fluid intake and weight form important components of management of heart failure (1). A typical Western diet contains around 6-10g of salt per day, yet heart failure patients should be limited to 1g per day. This disparity can be reduced by simply excluding salt rich foods and salt added at the table. Milk, cheese, bread, cereals, and some fresh vegetables (including spinach) must be eliminated in order to maximize the benefit from diuretic therapy. Fluid intake should be restricted particularly in patient with NYHA class IV because excessive fluid retention in the body is responsible for some of the clinical manifestations of heart failure. Alongside this aspect, body weight should be monitored regularly to detect fluid retention before it becomes symptomatic. Exercise on the other hand should be regularly carried out. Restriction of activity promotes physical deconditioning that may adversely affect clinical status and contribute to exercise intolerance. In a meta-analysis on the role of exercise training in HF , *Coats et al* showed that the benefits were comparable to those of pharmacotherapy; besides being additive to those of ACEIs and Beta-blockers (44). Finally, regular follow up of patients at out-patient clinics is crucial not only in confirming compliance but also in emphasizing the health education messages. *Tsutsui et al* in Japan found poor follow up to be the most important predictor of re-admission for patients with HF (as compared to previous number of admissions for HF, longer hospital stay, co-morbidities and unemployment) (45).

Pharmacotherapy for most patients with symptomatic left ventricular dysfunction should include combination of four drugs: a diuretic, an ACE inhibitor, a beta blocker and (usually) digitalis (1). The value of these drugs has been established in numerous large scale clinical trials. Patients with evidence of fluid retention should receive a diuretic

until a euvolemic state is achieved, and diuretic therapy should be continued to prevent recurrence of fluid retention. Even if a patient has responded favourably to a diuretic, an ACE inhibitor and a beta blocker should be initiated and maintained in patients who can tolerate them because they have been shown to favourably influence the long term prognosis of heart failure. Therapy with digoxin may be initiated at any time to reduce symptoms and enhance exercise tolerance. Spironolactone on the other hand should be reserved for patients with recent or current class III and IV symptoms, preserved renal function and a normal potassium concentration (1).

In end-stage heart failure, intravenous inotropes may be useful in palliation. Biventricular pacemakers and implantable defibrillators are also useful especially in patients awaiting heart transplant. Mechanical devices such as intra-aortic balloon pumps are also being tried but their mortality benefit has not been evaluated in randomized clinical trials (1).

3. JUSTIFICATION OF THE STUDY

Heart failure is a common leading cause of both morbidity and mortality worldwide. Anaemia, which has been shown to be a common problem in HF, is easy to diagnose and correct in most clinical settings. Facilities offering plain X- Rays are widespread across the country (unlike echocardiography) hence the need to re-define the role of a CXR in diagnosis of HF in our patients. Besides, this topic had not been researched on for many years in the country at large; last study having been done 14 years ago. In the aforementioned circumstances a study that endeavoured to define the current socio-demographics and clinical status of our heart failure patients on admission would answer pertinent questions on the broad topic of heart failure in the region.

4. RESEARCH QUESTION

What is the magnitude and profile of patients with acute decompensated heart failure at KNH and how are they disposed?

5. OBJECTIVES

Broad objective

To determine the epidemiology, clinical profile, selected laboratory features and non pharmacological therapy of heart failure patients admitted at Kenyatta National Hospital Medical Wards.

Specific objectives

1 To determine the six month period prevalence of acute decompensated heart failure in medical in-patients at KNH.

2 To determine the socio-demographic features of these patients.

3 To determine their baseline clinical, laboratory and radiographic profile: Blood pressure, Heart rate, Respiratory rate, NYHA functional class, Creatinine/electrolytes, haemoglobin level and chest X Ray features.

4 To asses the health education offered to these patients in terms of compliance to both drugs and out patient clinics, cessation of alcohol and smoking; and non-pharmacological instructions namely exercise, restriction of salt/fluid intake and weight monitoring.

6. METHODOLOGY

6.1 Site

Medical wards at Kenyatta National Hospital; Nairobi Kenya.

6.2 Population

Consecutive medical ward admissions with acute decompensated heart failure.

6.3 Design

Prospective clinical observational register of patients with acute decompensated heart failure.

6.4 Case definition

Patients admitted for presumed heart failure or whose chief complaint was dyspnoea and who satisfied the ***Modified Framingham clinical criteria for the diagnosis of Heart Failure***.

6.5 Inclusion criteria

- Age 13 years and above.
- Established diagnosis based on the Modified Framingham Clinical Criteria for the Diagnosis of Heart Failure.
- Informed written consent or assent

6.6 Exclusion criteria

- Patients who declined consent
- Recent (less than three months) or on-going myocardial infarction.

6.7 Sampling

Consecutive sampling technique was utilized.

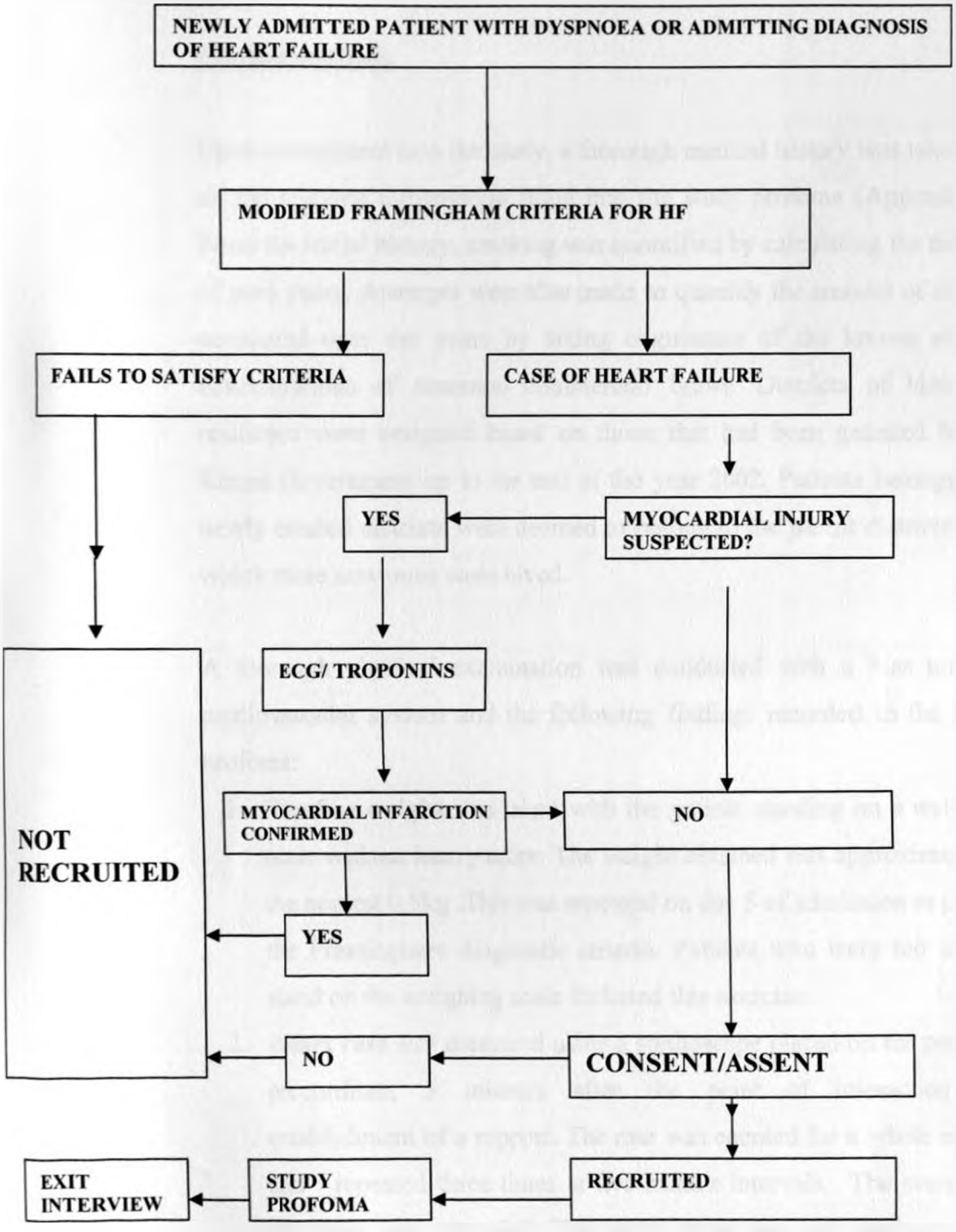
Screening and recruitment

On every post-admission day, the principal investigator with the assistance of a study assistant reviewed records of patients admitted with dyspnoea as one of the chief complains or whose admission diagnosis was heart failure. After establishing a rapport with the patient, a thorough medical history was taken and physical examination with a bias towards the cardiovascular system conducted. The ***Modified Framingham clinical Criteria for Diagnosis of Heart Failure*** was then administered. Those who satisfied the criteria were labelled as ***cases*** of heart failure. Cases with history suggestive of acute coronary syndrome underwent a 12 lead Electrocardiogram (E.C.G) plus serum cardiac troponins (if doubt still existed) to rule out recent or on- going myocardial infarction. Written consent (and assent where applicable) was sought from each of the cases after explanation of the objectives of the study in lay terms to them. Consenting/ assenting patients underwent the relevant laboratory and radiological investigations upon recruitment. Those who declined consent were not discriminated against in any way but left to undergo the routine ward evaluation and management. Recruited patients were followed up on alternate days until discharge/death.

6.8 **Sample Size Issues**

- This was a period limited study designed to run for exactly six months.
- Averagely, fifty patients are admitted into Kenyatta National Hospital medical wards per day.
- Over a six month period, approximately 9,000 (50 X 30 X 6) patients were expected to be admitted.
- Working on an approximate prevalence of 4% (1993 study prevalence 3.3%) about 360 patients were expected to be recruited into the study.
- Assuming a hospital mortality of about 20%, approximately 290 patients were expected be disposed thereby providing sufficient sample size for analysis.

6.9 Screening and study flow chart



6.10 Data collection

Clinical methods

Upon recruitment into the study, a thorough medical history was taken and all the relevant information filled into the study profoma (Appendix 5). From the social history, smoking was quantified by calculating the number of pack years. Attempts were also made to quantify the amount of alcohol consumed over the years by taking cognisance of the known alcohol concentrations of common commercial brews. Districts of birth and residence were assigned based on those that had been gazetted by the Kenya Government up to the end of the year 2002. Patients belonging to newly created districts were deemed to belong to the parent districts from which these new ones were hived.

A thorough physical examination was conducted with a bias towards cardiovascular system and the following findings recorded in the study profoma:

1. **Baseline weight** was taken with the patient standing on a weighing scale without heavy attire. The weight obtained was approximated to the nearest 0.5kg .This was repeated on day 5 of admission as part of the Framingham diagnostic criteria. Patients who were too sick to stand on the weighing scale forfeited this exercise.
2. **Heart rate** was measured using a stethoscope placed on the patients' precordium, 5 minutes after the point of interaction and establishment of a rapport. The rate was counted for a whole minute and repeated three times at five minute intervals. The average of the three was recorded. The radial pulse was not used as it can unreliably underestimate the heart rate especially in the setting of atrial fibrillation.

3. **Blood pressure** was measured using a standard validated mercury sphygmomanometer (cuff length 35 centimetres, width 12centimetres) with the patient lying supine and propped up in bed with arms at the same horizontal level as the heart. Initially, to take care of patients with peripheral occlusive arterial disease, the pressure was taken from both arms. From the arm with highest reading, three readings at five minute intervals were taken and their average recorded.

4. **Leg Oedema** was deemed to be present if a patient had swollen legs and had demonstrable pitting upon application of gentle but firm pressure at the level of the ankle.

5. **Jugular venous pressure** was measured by taking a perpendicular line from the sternal angle to the highest point of visible internal jugular pulsations within the neck with the patient propped up in bed at an angle of 45^{0}.

6. **Apex beat** was taken as the lowest and outermost point of definite cardiac impulse. It was deemed to be displaced if it was found in any other position other than the fifth intercostals space within the mid-clavicular line.

7. **Pulmonary rales** were demonstrated by listening at the lung bases for soft crackles with patient propped up in bed.

8. **Liver span** was measured along the right mid-clavicular line using a tape measure after demarcation of its margins by palpation, percussion or ballotment (whichever was applicable). Hepatomegally was deemed to present if the measured span was in excess of fifteen centimetres.

9. **Ascites** was deemed to be present if a fluid thrill was positive or shifting dullness was demonstrated in the (distended) abdomen.

The patients were then followed up on alternate days until discharge or death. Two weeks after admission (or upon discharge whichever came earlier), another interview was conducted by the PI to collect information received on health education and non-pharmacological instructions. Aspects of health education of interest included information on cessation of smoking, alcohol use, regular clinic attendance and drug compliance. Non pharmacological instructions of interest included need for regular exercise, reduction of both salt and fluid intake and weight monitoring. Open ended questions for each aspect with regard to HF were used to obtain information from each patient.

For quality control, all clinical procedures including the exit interview were conducted by the PI. The SA only assisted in the initial file/ patient reviews and identification of patients due for discharge.

Laboratory methods

Two millilitres of venous blood was collected into EDTA anticoagulated vacutainer from every recruited patient for determination of haemogram. The test was done by a qualified technician at the Department of Haematology (KNH). For standardisation of results, all the tests were run using one machine – ***CLL- DYN 3200***. From these results, the haemoglobin level and the mean corpuscular volume (MCV) were recorded into the study profoma.

A further two millilitres of venous blood was collected into a plain vacutainer for determination of serum creatinine and electrolytes. The tests were run by a qualified technician at the Department of Biochemistry (KNH) using ***Olympus***

AU – 640 machine. From the results the serum creatinine, sodium and potassium were recorded into the study profoma.

Imaging studies

A plain chest radiograph was done for each patient within 24 hours of admission. Every effort was made to take a standard good quality posterior/ anterior (PA) view. All films were taken at the KNH X–Ray Department using a specific machine: ***Medio 50 CP Model 14490206001003.*** Interpretation of the films was undertaken by a consultant radiologist on duty blinded to the patient's clinical state. Interpretation of AP views was conducted with caution with respect to the cardiothoracic ratio (CTR); choosing to 'eyeball' for evidence of gross cardiomegally. Cephalization of pulmonary vessels, Kerley B lines, alveolar oedema and pleural effusion were the other radiological features of interest. No attempts were made to correct for intra or inter-observer variations.

Echocardiographic data - though not strictly a study directed activity - was collected when available (particularly the Left ventricular Ejection Fraction-LVEF), to add more objective evidence to the clinical diagnosis.

6.11 Statistical analysis

Data prospectively recorded into the study profoma was entered into computerized data entry sheets. Cleaning and verification was done on weekly basis to ensure validation and completeness of the information. Statistical analysis was performed using the statistical package for social sciences version11.2 software for windows.

Descriptive statistics such as frequency, percentages, proportions, mean, median and mode were used for most of the data. The student t-test was used to compare means of two variables, while the ANOVA was used in the comparison of means of more than two variables. Data was presented in form of tables, pie-charts and graphs.

The level of significance was set at $p < 0.05$, and a 95 % confidence interval was applied to the numerical variables that were normally distributed.

7. ETHICAL CONSIDERATIONS

1. The study was conducted only after approval by the Department of Clinical Medicine and Therapeutics of the University of Nairobi; and the Kenyatta National Hospital Ethics and Research Committee.

2. Before recruitment into the study all eligible patients received information concerning the purpose of the study in lay terms and a signed consent obtained. For those under the age of 18 years, an assent was also sought.

3. Recruited patients were free to withdraw from the study at any time of the study period without being discriminated against in any way.

4. The investigations carried out on the patients were relevant based on the current scientific understanding of the pathophysiology and risk stratification of patients with acute decompensated heart failure.

5. Confidentiality was maintained by excluding patients' names on the computerized entry sheets and storing the profomas in a secure location.

6. Results were communicated back to the patients plus their medical teams.

7. Gaps noted in the patients' knowledge on health education pertaining to their comprehensive care were filled during the exit interview.

8. RESULTS

This was a period limited study conducted over six months: 5/12/2007 to 4/6/2008 both dates inclusive. During the period, a total of 5043 patients were admitted into the medical wards. Of these, 458 were screened for heart failure using the '*Modified Framingham Criteria for diagnosis of Congestive heart failure*'. Two hundred and eighty six patients were finally recruited into the study after meeting the inclusion criteria. This gave a prevalence of 5.7%.

The details of monthly admissions were as follows

Table 1: Monthly admissions and gender specific recruitment rates of heart failure patients

MONTH	TOTAL ADMISSION	FEMALES RECRUITED	MALES RECRIUTED	TOTAL RECRUITMENT
5/12/2007-4/1/2008	822	24 (54.5%)	20 (45.6%)	44
5/1/2008-4/2/2008	840	17 (39.5%)	26 (60.5%)	43
5/2/2008-4/3/2008	736	32 (61.5%)	20 (38.5%)	52
5/3/2008-4/4/2008	847	22 (47.8%)	24 (52.2%)	46
5/4/2008-4/5/2008	839	24 (53.3%)	22 (46.7%)	46
5/5/2008-4/6/2008	941	35 (62.5%)	20 (46.7%)	55
GRAND TOTAL	5043	154 (53.8%)	132 (46.2%)	286

8.1 Socio-demographic profile

Sex

Of the 286 patients recruited, females comprised 154 and males 132 patients.

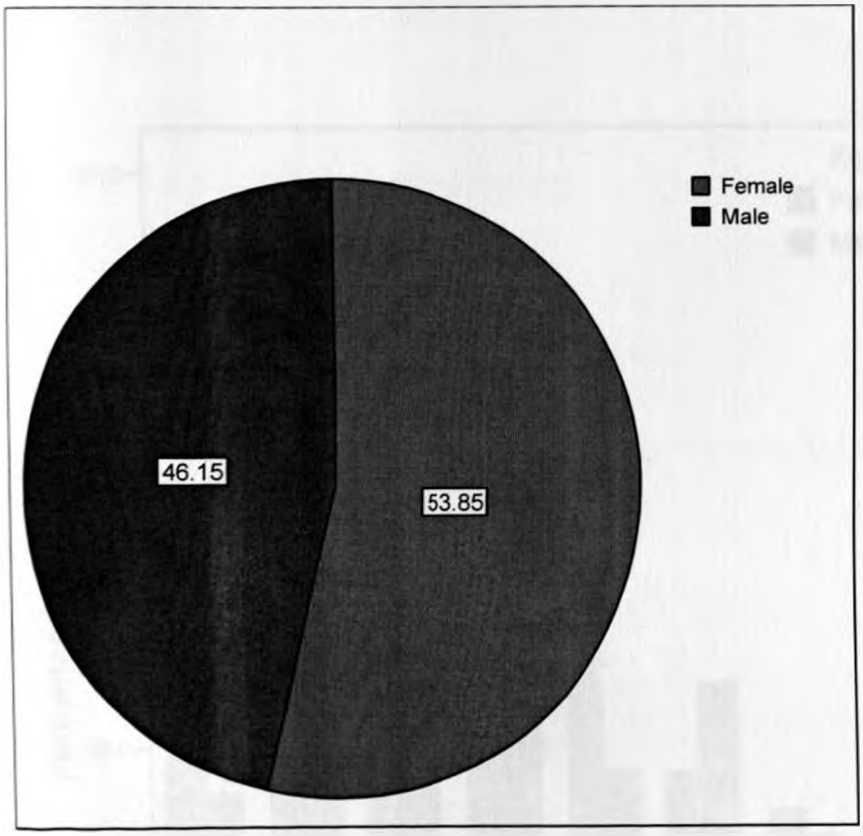


Figure 1 :Sex distribution of heart failure patients

Age

The mean age of the patients was 44 years. When stratified for sex, females had a mean age of 45.2 years and males had a mean of 42.4 years .This difference did not however reach statistical difference ($p = 0.258$; $t = 1.133$). The peak age group at presentation was between 20 to 30 years. A quarter of the patients were elderly (60 years and above). Patient distribution by sex in all age groups was similar except in the 20 – 29 age groups where there was a preponderance of male patients.

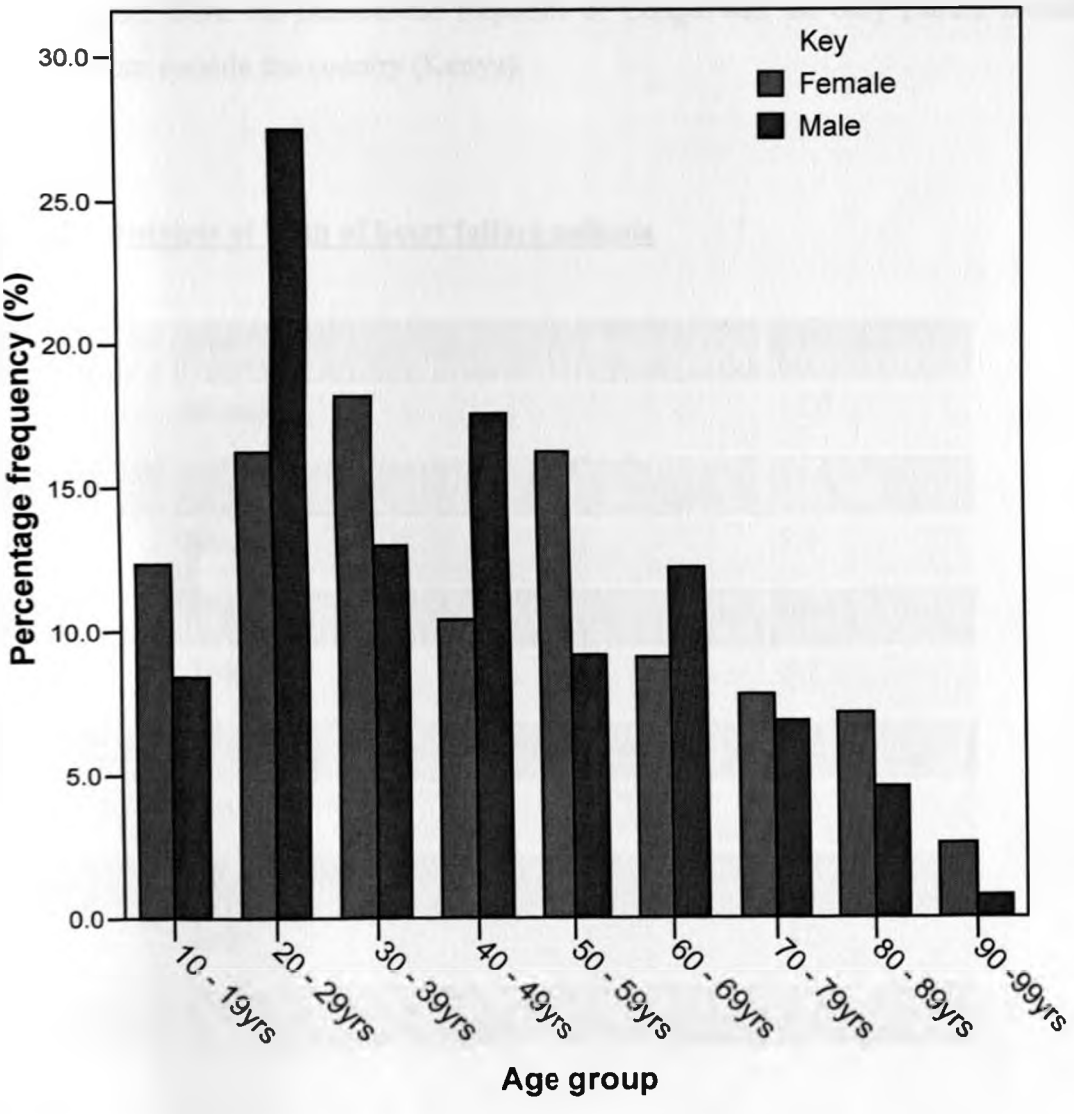


Figure 2: Age and sex distribution of heart failure patients

District of birth

Majority of the patients were born within Central Kenya districts and Nairobi. Thus Murang'a, Kiambu, Thika, Nyeri and Nairobi accounted for more than 50 % of the patients' districts of birth. Few pockets outside these regions were noted in Kisii, Kisumu, Machakos and Nyandarua Districts. Western, Rift Valley, Coast and North Eastern (provincial) districts were poorly represented.

One foreigner from the Democratic Republic of Congo was the only patient recruited who was born outside the country (Kenya).

Table 2: Districts of birth of heart failure patients

No	District	percentage
1	Murang'a	14.0
2	Kiambu	11.5
3	Nairobi	8.4
4	Machakos	6.6
5	Thika	5.2
6	Meru	4.9
7	Nyeri	4.5
7	Nyandarua	4.5
9	kisii	4.2
10	Others	5.9

District of residence within past five years

This reflected the pattern of districts of birth. Nairobi however was home to almost a third of all heart failure patients admitted.

Table 3:Districts of residence of heart failure patients within past 5 yrs

No	District	Percentage
1	Nairobi	30.8
2	Kiambu	10.2
3	Murang'a	8.2
4	Machakos	5.6
5	Kisumu	4.9
6	Thika	4.9
7	Meru	4.2
7	Nyandarua	4.2
9	Kisii	3.8
10	Others	4.2

Formal education level

Majority of patients were poorly educated. Fifteen percent of the patients had no formal education. A further 56.7% and 24.6% had attained primary and secondary education respectively. Only 3.5% of them had attained post-secondary education.

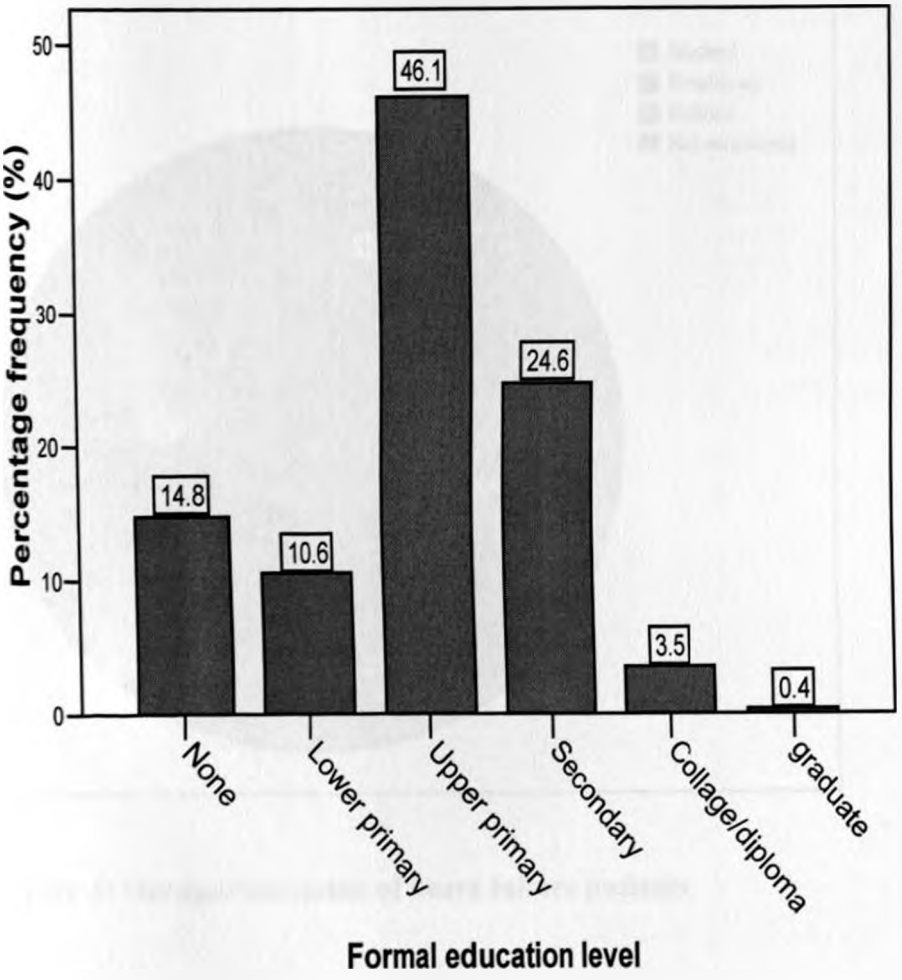


Figure 3: Level of education of heart failure patients

Occupational status

Forty seven percent of the patients were not and had never been engaged in any gainful employment. About forty percent were actively engaged in some form of employment (self or formal) while 10.1% were students. The remaining 2.8% of the patients were retirees.

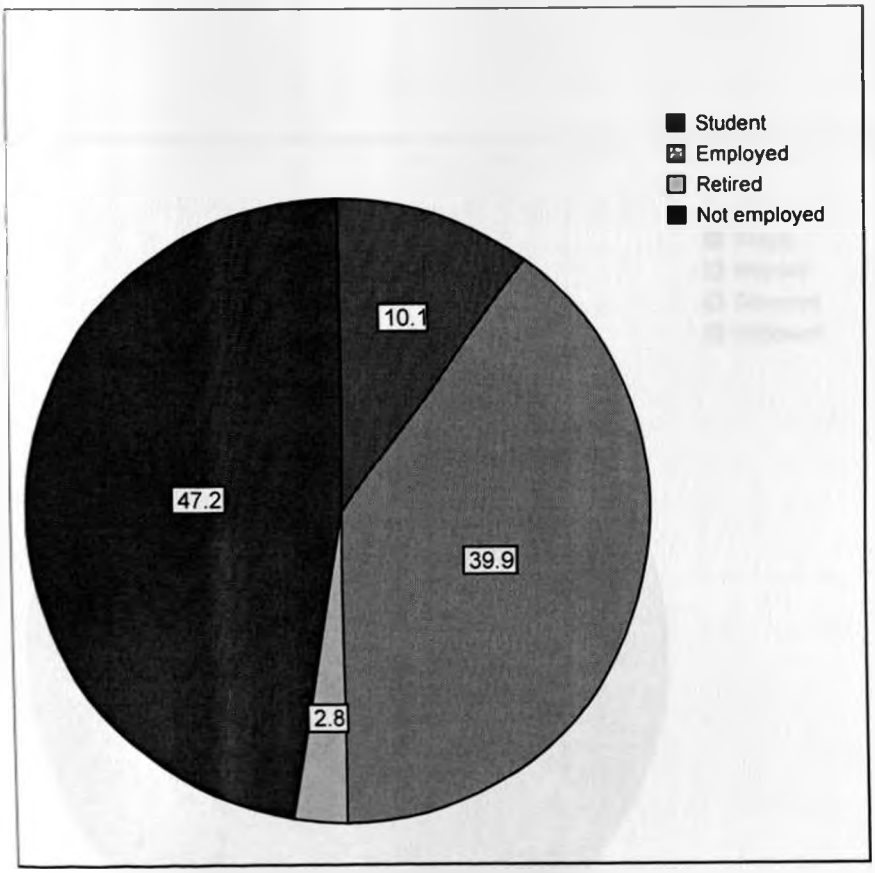


Figure 4: Occupation status of heart failure patients

Marital status

One hundred and fifty three (53.4%) patients were married. Of the remainder, 33.9% were single, 8.4% were divorced and 3.5% were widowed.

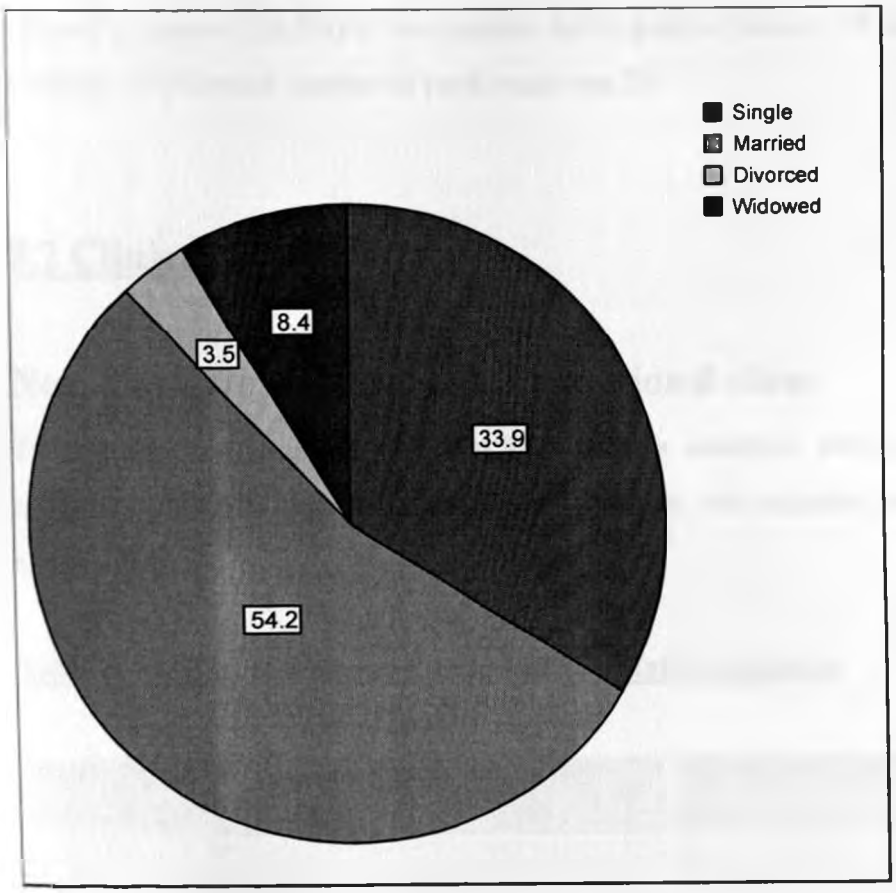


Figure 5: Marital status of heart failure patients

Alcohol consumption

Two hundred and four (71.3 %) patients had no history of alcohol consumption. For the less than one third with such history (past or current), quantification of the amount of alcohol consumed was difficult because the majority consumed local brews which do not have standardized alcohol content.

Tobacco smoking habits

Almost a quarter (23.2%) of the patients had a positive history of either past or current smoking. Their mean number of pack years was 20.

8.2 Clinical profile

New York Heart Association functional class

Two hundred and seventy two (94.8%) patients admitted with moderate to severe symptoms (NYHA classes III and IV). No patient was admitted with NYHA class I symptoms.

Table 4: NYHA class at presentation of heart failure patients

NYHA FUNCTIONAL CLASS	PERCENTAGE
1	0
II	5.8
III	41.8
IV	52.4

Haemodynamics

The mean systolic blood pressure of the patients was 109mmHg while the mean diastolic blood pressure was 69mmHg. Those with significantly elevated blood pressures were 12.8% (systolic BP above 140mmHg) while those with significantly depressed systolic blood pressures were 19% (systolic blood pressure of less than 90mmHg). Fifteen percent had diastolic hypertension (BP >90mmHg) while 22.7% had diastolic hypotension (BP <50mmHg).

The mean heart rate was 105 beats per minute. This is in the tachycardia range but not significantly elevated compared to the Framingham criteria (more than 120beats/minute).The number of patients with significant tachycardia was 81 (28.2%). None of the patients had significant bradycardia (heart rate of less than 50 beats/minute)

Respiratory rate

The mean respiratory rate was 27 breaths per minute. All patients were tachypneic. Thirty one percent had a respiratory rate above 30 breaths per minute.

Other clinical parameters from history

The most common cardiac symptoms at presentation were dyspnoea, easy fatiquability, palpitations, night cough and orthopnea. History of shortness of breath and easy fatiquability were almost invariable findings. The five commonest symptoms cited were present in more than 90% of patients at presentation. Other important symptoms included history of leg swelling at 80%, and abdominal swelling at 74%. Sixty percent of patients had a history of past admission for heart failure either at KNH or at peripheral hospitals before referral.

Table 5: Frequency of presenting symptoms of heart failure patients

SYMPTOM	FREQUENCY
Shortness of breath	100%
Easy fatiquability	99%
Palpitations	97%
Night cough	93%
Orthopnea	96%

Clinical signs

A raised jugular venous pressure (JVP) was the commonest clinical sign at 83.8%, followed by tender hepatomegally at 74.4%. Bilateral leg oedema was present in 73% of the patients while cardiac murmurs were present in only 58.55 of the patients.

Table 6: Physical signs at presentation of heart failure patients

SIGN	FREQUENCY
Raised JVP	83.8%
Tender hepatomegally	74.4%
Bilateral leg oedema	73%
Basal rales	60%
Murmurs	58.5%
Ascites	50%
Displaced apex	48%

8.3 Laboratory parameters

Haemoglobin

The mean haemoglobin level was 12.92g/dl while the mean MCV was 85.6fl. When stratified by sex the mean haemoglobin level for female patients was 12.7g/dl and 13.6g/dl for male patients.

Renal function and electrolytes

Creatinine

The mean serum creatinine concentration was 155.8micromlo/l with a median of 97micromol/l. Twenty seven percent of the patients had deranged renal function.

Sodium (Na+)

The mean serum sodium concentration was 137mmol/L. The percentage of patients with serum sodium values within the reference range was 56.8%. Hyponatraemia was present in 35.6% of the patients while 7.4% had hypernatraemia.

Potassium (K+)

The mean serum potassium was 4.8mmol/L. Most patients' readings fell within the reference range – (i.e. 89.7% of the patients). Hypokalaemia and hyperkalaemia were present in 7.4% and 2.9% of the patients respectively.

Table 7: Selected laboratory findings at presentation of heart failure patients

Measurement	Mean	SD	median	mode
Hb in g/dl	12.96	2.4	13.3	14.2
MCV in fl	85.67	1.7	86.0	86.0
Creatinine	155.18	276.7	97.0	89
Na+	137.21	6.6	137.4	132
K+	4.38	0.66	4.3	4.2

8.4 Chest Radiographic features

Gross cardiomegally was the commonest radiological finding at 97.3% while Kerly B lines were rare at 12%.

Table 8:CXR features at presentation of heart failure patients

FEATURE	FREQUENCY
Cardiomegally	97.3%
Alveolar oedema	81.4%
Cephalization	68%
Pleural effusion	34 %
Kerly B lines	12.%

8.5 Disposition

Generally, very little information was given to the patients concerning their comprehensive treatment before disposition.

Table 9: Disposition of heart failure patients

INSTRUCTION/ASPECT	PERCENTAGE RECEIVED
Follow up at Cardiac/OP clinic	79.6%
Need for drug compliance	64%
Reduction of salt intake	17%
Reduction of fluid intake	12.6%
Need to avoid /Quit smoking	14%
Need to avoid /quit alcohol	9.1%
Role of exercise	7%
Weight monitoring	3%

8.6 Outcome

The mean length of hospital was 6.84 days with a range of 2 to 27 days.

Thirty one patients died while undergoing treatment giving an in-hospital mortality of 10.8 %.

9. DISCUSSION

The prevalence of acute decompensated heart failure in this study was 5.7% .This prevalence is very high considering that non communicable diseases account for less than half of all medical admissions – the other half comprising of infectious diseases including human immunodeficiency virus (HIV) co-morbidities (46). The figure has almost doubled compared to the findings in a study conducted by *Oyoo* et *al* in the same hospital 1993 (4). The difference could be explained by the design and duration of the two studies or the changing pattern of cardiovascular diseases in the country over the past fifteen years. This study was carried out as a registry over a six month period whereby virtually all patients who satisfied the criteria for inclusion were recruited. The earlier study had a fixed sample size and was carried out over a three months period. On the other hand, the aetiology of heart failure was not addressed in this study as it was not one of our objectives and comment on the same is to say the least, speculative.

Regionally, *Obineche et al* found a similar prevalence of 10.6% at Lusaka Teaching Hospital in Zambia in 1976 (47). A recent population based survey in Mozambique specifically looking at *Endomyocardial fibrosis* (a form of cardiomyopathy endemic in some parts of Africa) found a prevalence of 19.8%; though only 48% of them were symptomatic for heart failure (48). In the western populations, the overall prevalence of heart failure in UK hospitals for example, stands at 5% with a population prevalence of 0.4 – 2% (49)

The mean age of the patients was 44 years. When stratified by sex the mean age for men was 42 years while that for females was 45.2 years. This difference however did not reach statistical significance. The figures compare well with findings from two Ghanaian studies: the first one published in the year 2000 by *Amoah et al* that reported a mean age of 44 years and a more recent one by *Owuso et al* in 2007 that reported a mean age of 53.9 years (16,18). In the developed societies, heart failure is a disease of the elderly. The average age at presentation in United Kingdom is 76 years; while population surveys

show that more than 6% of persons aged above 65 years are affected (19). In the USA, more than 80% of heart failure patients are aged above 65 years (6). This age difference could be due to differences in the aetiology of the condition between the two populations: ischemic heart failure in the western societies while cardiomyopathies and rheumatic heart disease in Africa. Rheumatic heart disease is epidemiologically known to affect young age groups in poverty stricken areas such as Sub Saharan Africa (17). Cardiomyopathies are also known to be endemic in Africa and tend to be a disease of middle age (17). Conversely, ischemic heart disease tends to occur in affluent and elderly populations (8). Another possible explanation could be the higher life expectancy enjoyed by the western societies which is in seventies as opposed to 48 years in Kenya (50)

Sex distribution of patients was slightly in favour of females though not statistically significant (53.8: 46.2%). A similar finding was reported by *Oyoo et al.* In the USA the ADHERE registry reported similar margins as our data with female to male ratio of 53: 47% (51).

The majority (86%) of the patients studied had been residing within Nairobi and surrounding districts mainly from Central and Eastern Provinces. Outside this zone, significant areas of residence included Kisumu, Nyandarua and Kisii Districts. This is most likely due to distance, because it is easier patients residing within Nairobi and its environs to access health care at KNH. When compared with overall hospital medical admissions over the same period, residents of Nairobi province (considered as a single district in this study) took up 76.5% of the bed capacity at the hospital (46). In contrast, only 31.8% of the heart failure patients were actually residents of this city. This could be due the fact that heart failure patients are referred for specialised care from far away districts and therefore comprises a larger majority of (68.2%) patients at the hospital. Kajiado District, whose northern divisions actually form part of the southern suburbs of Nairobi city, is not represented at all in our results but becomes second at 5% in terms of overall hospital medical admissions. Residents of Murang'a district who comprised 8.7% of the patients studied accounted for a paltry 1.7% of the overall medical admissions; meaning that the district bears a significant burden of heart failure morbidity. The reasons

behind this observation are unclear. Another significant finding is the finding of Kisumu district's conspicuous position at position 5 among the patients' district of residence whilst it does not appear within the top ten districts of birth. The reasons behind this finding are unclear.

On the basis of education, 70% of the patients had attained only primary formal education or none at all: a reflection of their overall low socio-economic status. Similarly, about 60% of the patients had no form of income. The earlier study carried out by *Oyoo et al* registered worse statistics on this issue; with more than 80% of the patients having attained none or only primary education. A possible explanation could be the fact that heart failure patients of higher socio-economic status do seek medical treatment at private hospitals due to affordability leaving the publicly owned KNH to people from humble economic backgrounds. The clinical implication of this finding is that most of the HF patients may not be able to afford quality healthcare in terms of diagnostic work-up and drugs making compliance difficult. Likewise understanding health education and non-pharmacological information plus their implications on their overall outcome may be difficult. The patients understanding of these messages and total co-operation is indispensable in terms of reducing mortality and morbidity in HF. *Tsutsui et el* in Japan looking at socio-environmental predictors of hospital re-admissions for heart failure identified lack of occupation as one of the important factors with an odds ratio of 2.5 (with 95% CI) (45).

Social support, especially marriage is important in overall management of heart failure. 53% of the patents were married at the time of the study. This figure is remarkably high given that 32% of the patients were below 30 years. Western literature has scanty information on this aspect.

Smoking and excessive alcohol consumption are known to be some of the traditional risk factors for cardiovascular disease: alcohol for secondary dilated cardiomyopathy and smoking for coronary heart disease. Twenty nine percent of our patients had a positive history of alcohol consumption. Consumption of more than 90 g/day of alcohol for more

than ten years is generally considered significant for the development of cardiomyopathy. Quantification of the amount consumed over the years was difficult because most of the patients consumed local brews with no standard alcohol content. *Samia et al* studying the role of physical activity in heart failure reported a 52% prevalence of regular alcohol consumption among his patients (52). The study did not however discuss further the role of alcohol in the aetiology of their heart failure nor its implications on management. On the other hand, 23.2% of our patients had history of past or current smoking. On average they were heavy smokers with a mean of 20 pack years. The prevalence of smoking is apparently rising in our population. For instance, *Ogola et* al studying cardiovascular risk profile among hypertensive patients at KNH reported a prevalence of 6.5 % ten years ago (53). *Mohammed et* al also looking at cardiovascular risk factors and target organ damage in diabetic out-patients seen at KNH in 2002 reported a prevalence of 7% (54). This is most likely due to massive advertisement campaigns targeting the youth in third world by tobacco companies. The prevalence of smoking in heart failure patients in the western countries is much lower than in our set up; in the ADHERE registry for example, only 14% of the patients report such history (55). This is again a reflection of heavy public campaigns against smoking undertaken by the western governments. In contrast, worse statistics on cigarette smoking are reported in the Arab world with Egyptian data showing that 81% of its male patients with heart failure are either pasts or current smokers (56).

Clinically, most patients were admitted with moderate to severe symptoms. Thus, 94.2 % of the patients were in NYHA functional classes III and IV. *Oyoo et al* found a similar margin in 1993. A similar pattern of clinical presentation was reported in a recent study from Ghana (18). The reason behind this trend is most likely due to the fact that only very sick and unstable patients are admitted in our hospitals due to overstretched health facilities. Fairly stable patients seen at the emergency department are usually put on treatment and booked for follow up at the specialist cardiac clinic.

Table 10:Comparison of Clinical severity of heart failure patients

NYHA class	Barasa Kenya (2008) N= 286	Oyoo Kenya (1999) N= 91	Kofi Ghana (2007) N=100
I (%)	0	0	0
II (%)	5.8	5.5	6
III (%)	41.8	31.9	29
IV (%)	52.4	52.6	65

Haemodynamically, the mean heart rate of the patients was 105 beats/minute.

This was lower than what is considered significant (*in the Modified Framingham Criteria)* to constitute a minor criterion for heart failure diagnosis. McAlister et al in a specialised out- patient setting in Ottawa Canada found a mean heart rate of 80 beats per minute (23). The difference between the two could be due to different settings in the two studies; in-patient setting for our study where only very sick patients are admitted versus out-patient setting in the Canadian study where all forms of HF severity patients were recruited. Our data showed a mean systolic BP of 109 mmHg and diastolic BP of 69mmHg. *Fonarow* et al looking at risk stratification for in-hospital mortality in patients admitted with acutely decompensated heart failure in the ADHERE registry showed that systolic BP of less than 115mmHg was associated with adverse outcomes while patients with systolic BP of more than 160mmHg had the best outcomes (57). Our data however was not statistically powered to detect a significant statistical significance between Blood Pressures and mortality. The OPTIMIZE-HF registry whose mortality rates are much

lower than ours reported a mean Blood pressure of 142mmHg (systolic) and 76mmHg (diastolic) (58). The difference between blood pressures in our study and that of the OPTIMIZE- HF registry could be due to the fact that our patients are admitted when they are more sick and therefore experiencing more haemodynamic decompensation.

Majority of the patients had a normal haemoglobin level. The mean Hb was 12.9g /dl. This may be due to the fact that most of our patients did not suffer from nutritional deficiencies or significant co-morbidities. This compares well with the ADHERE registry where a mean Hb of 12.2mmHg was reported (51).

Our data revealed that a significant proportion of our patients had deranged renal function. Twenty seven percent of patients had their creatinine level above the normal reference value while the mean value was 156micromol/l. Significantly deranged renal function at baseline has been associated with adverse hospital outcomes (57). The ADHERE registry recorded worse figures with a mean of 183micromol/l for African Americans and 140.8micromol/l for white patients (51). The OPTIMIZE - HF registry is no better with a mean creatinine of 158micromol/l in a recent publication by *Fonarow* et al (57).

Among the electrolytes, significant derangement was seen with serum sodium concentration. More than a third (35.8%) of the patients were hyponatriaemic but their mean fell within the reference range. In the OPTIMIZE HF registry, the mean sodium concentration of the patients was 138mmol/l; with 19.7% of the patients being hyponatraemic (58). The study showed that hyponatraema was associated with a 19% risk of in- hospital death (after correcting for other traditional predictors of adverse outcomes.). In both studies, it is worth noting that patients with hyponatraema were clinically similar with their normonatraemic counterparts in terms of age, gender and NYHA functional class. Hypokalaemia on the other hand was less common, affecting only 7.4% of the patients. The mean serum potassium concentration also felt within the reference range at 4.8mmol/l.Hyperkalemia was even less common; affecting less than 3% of the patients. Clinical implication of this finding is that the larger majority of heart

failure patients can receive ACEIs and spironolactone without worrying about their hyperkalaemia adverse effects although monitoring is obviously mandatory. Studies for comparison on potassium ion handling in heart failure are scanty in most of the literature reviewed.

Radiological findings also revealed crucial data in heart failure patients. Gross cardiomegally and alveolar oedema on plain chest radiographs were very sensitive with frequencies of 97.3% and 81.4% respectively. These two radiological parameters become even more important because they form part of the major criteria in the *Modified Framingham Clinical Criteria for diagnosis of heart failure*. Recent data published based on ADHERE registry found a prevalence of alveolar oedema in hospitalised patients with acute decompensated heart failure to the tune of 81%; consistent with our data (55). The Clinical implications of these findings are that a plain CXR is still a crucial investigative tool in the diagnosis of HF particularly in peripheral hospitals where echocardiography is not widely accessible.

Our data reveals that patient disposition was not well conducted. No proper focussed information was offered to the patients concerning neither their syndrome nor the need for their co-operation to ensure a successful treatment programme. As an example, smoking and alcohol consumption, which could be aetiologically related to their condition, was reported in about a quarter of the patients yet only 14% and 10% of them respectively ever received information on their cessation. Information on weight monitoring was received by only 3% of the patients and yet such information is a simple surrogate marker which the patients themselves can cage their adherence to fluid restriction, reduction of salt intake and pharmacotherapy. In fact, most of the patients become symptomatic when their weight gain is in excess of five kilograms. Similarly, information on the role of exercise was offered to only 7% of the people. The concept of non- pharmacological management of heart failure has apparently not taken root among the medical fraternity across the board as evidenced from another study from Ghana where 73% of patients admitted with acute decompensated heart failure in a university teaching hospital never received any form of non pharmacological therapy (18). A recent

publication from USA showed that a structured physical exercise programme over a twelve week period can improve LVEF by 35% and reduce pro-BNP serum levels by 40% (59). These figures are far beyond what is achieved by the standard life prolongation drugs in heart failure which together in combination (B- blockers and ACEIs) improve LVEF by 12% over a three year period. A recent publication based on the OPTIMIZE-HF registry showed that non-adherence to both medication and diet were some of the commonest factors precipitating hospital admission for acute heart failure (54). Indeed, the OPTIMIZE-HF registry is perhaps the only one with a comprehensive 'discharge therapy instrument' administered to its patients at the point of hospital departure.

Our in–hospital mortality of 10.7% was slightly lower than data from Ghana where their in–hospital mortality at the *Komfo Anokye Teaching Hospital* (KATH) was reported to be 15% in a recent study (44). However, it is much higher compared to data from the western countries. The EDHERE registry for example, recorded an in- hospital mortality of 2.1% for their African American patients and 4.5% for the white population (60). This could be due to the fact that KNH is a specialized tertiary centre admitting more severe cases unlike the ADHERE registry which consists of community based hospitals. The other reason could be due to the superior level of health care offered to the patients in the ADHERE registry.

SUMMARY OF RESULTS AND CONLUSION

Our data shows that the prevalence of acute decompensated heart failure at KNH was 5.7% of all medical admissions over the six month period. They registered a high mortality at 10.7%. Most of the patients come from the densely populated Central, Eastern and Nyanza provinces. The peak age at presentation was in the 20- 40 year age bracket with a mean age is 44yrs. Both males and females were equally affected. Most of them were in the low socio-economic bracket. Their clinical characteristics were similar to their western counterparts making current diagnostic tools appropriate for their evaluation. Widely available laboratory tests like haemogram, renal function tests and

plain radiography were useful both in diagnosis and baseline evaluation. Finally, non pharmacological modes of therapy were sub-optimally offered.

In conclusion, acute decompensated HF is a common medical problem at KNH with an equally high mortality. Majority of the patients are either young or middle aged and tend to present with severe disease. No clear information was given to them on health education and non- pharmacological aspects of heart failure treatment at disposition.

10. RECOMMENDATIONS

1. A larger registry comprising more patients from more hospitals and designed to last for a longer period could provide more information concerning these patients especially concerning risk stratification with regard to traditional risk factors for adverse outcomes: serum urea, creatinine and systolic BP at admission.

2. A study to look at post discharge outcomes at three, six months and up to a year could provide important information on their prognosis following hospitalization for heart failure.

3. A comprehensive tool encompassing all health education messages and non-pharmacological modes of therapy should be designed by the cardiology unit of KNH and administered to HF patients before discharge or written in a language they can understand as a leaflet and attached to their discharge summary.

11. STUDY LIMITATIONS

1. Kenyatta national Hospital is a tertiary referral centre and this may have created some bias in the severity of cases admitted. However the hospital serves as a primary care centre for Nairobi city and its environs with a population of 4.7 million people

2. The findings of this study can not be generalised to apply to patients admitted at non specialised primary care and other community based hospitals.

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APPENDIX 1

MODIFIED FRAMINGHAM CLINICAL CRITERIA FOR DIAGNOSIS OF HEART FAILURE

Major criteria

- Elevated jugular venous pressure.
- Pulmonary rales.
- Pulmonary oedema on chest X-ray
- Third heart sound.
- Orthopnea.
- Cardiomegally on chest X-ray
- Paroxysmal nocturnal dyspnea
- Weight loss of more than or equal to 4.5kg over 5 days in response to treatment of presumed heart failure

Minor criteria

- Bilateral leg oedema.
- Nocturnal cough.
- Dyspnoea on ordinary exertion.
- Hepatomegally.
- Pleural effusion.
- Tachycardia (≥ 120 bpm)
- Weight loss of more than or equal to 4.5kg over 5 days.

Diagnosis:

The diagnosis of heart failure requires that 2 major or 1 major and 2 minor criteria cannot be attributed to another medical condition.

From Senni, M, Tribouilloy, CM, Rodeheffer, RJ, et al, Circulation 1998; 98:2282; ada pted from McKee, PA, Castelli, WP, McNamara, PM, Kannel, WB. N Engl J Med 1971; 85:1441.

APPENDIX 2

SCREENING PROFOMA

Hospital No.....Ward---- Age: ----- Sex: -------Date------

Patient Status: Available..... Deceased.....

Tick appropriately if present, Cross if absent

History

Orthopnea*

Paroxysmal nocturnal dyspnoea*

Dyspnoea on ordinary exertion

Nocturnal cough

Physical Findings

Heart rate > 120beats/min

Bilateral leg oedema

Raised jugular venous pressure*

S3 gallop*

Pulmonary rales*

Tender Hepatomegally

CXR

Cardiomegally*

Pulmonary oedema*

Pleural effusion

At least 2 major or 1 major (*Italic with **) and 2 minor criteria cannot be attributed to another medical condition = Heart Failure

CASE	NOT CASE

APPENDIX 3

Ward.....

Room..... Date.....

ADULT CONSENT FORM (AGE 18YEARS AND ABOVE)

I, Dr **Barasa Ayub Felix**, a postgraduate student in the Department of Clinical Medicine and Therapeutics of the University of Nairobi, am conducting a study on heart failure patients. This is a non-interventional study looking at your demographics, clinical, laboratory and chest X-ray features; and non drug management you will receive in this hospital as a heart failure patient. Heart failure is in turn defined as a state in which your heart is not able to adequately pump enough blood to meet the needs of your body's tissues.

If you agree to participate in this study, I will take a full medical history from you and conduct a physical examination for purposes of confirming the diagnosis (of heart failure). I will also take 4ml of blood for determination of your blood counts and kidney function; and order for a chest X-ray to show me the size of your heart and presence of excessive fluid in your lungs. There will be minimal pain when drawing blood while the X-ray will expose you to minimal radiation.

The results of these investigations will be explained to you and a copy given to your ward doctor

Participation is voluntary and you are free to withdraw at any time during the course of this study period. Your refusal to participate or withdrawal from the study will not in anyway affect the quality of your treatment.

All the information obtained will be treated confidentially.

I ..... (Full name) of ..... understand the above and voluntarily accept to participate in the study

Signed.....Date.....

I confirm that I have explained to the patient the above statement

Signed ..... Date..... (Interviewer)

Contacts

Patient Tel.....Physical address.....

Next of kin/ caretakers:

1. Name.....Tel.....Relationship.....
2. Name.....Tel.....Relationship.....

APPENDIX 4 **Ward.....Room.....Date.....**

CONSENT FORM FOR MINORS (AGE 13- 17YEARS)

Investigator: Dr Barasa Ayub Felix

Position: Postgraduate student, Department of Clinical Medicine and Therapeutics, University of Nairobi.

Dear sir/ Madam,
I am conducting a research study on heart failure patients. This is a non-interventional study looking at your child's demographics, clinical, laboratory and chest X- Ray features; and the non- drug management your child will receive in this hospital as a heart failure patient. Heart failure is in turn defined as a state in which someone's heart is not able to adequately pump enough blood to meet the needs of his/her tissues.

If you agree to let your child participate in this study, I will take a full medical history and conduct a physical examination on him/her for purposes of confirming the diagnosis (of heart failure). I will also take 4ml of blood for determination of his/ her blood counts and kidney function; and order for a chest X-Ray to show me the size of his/ her heart and presence of excessive fluid in the lungs. There will be minimal pain when drawing blood while the X-ray will expose him/ her to minimal radiation. The results of these investigations will be explained to you and a copy given to your ward doctor

This study will determine if your child is receiving the (internationally recommended) standard non- drug management measures for heart failure in this hospital or not. If any gap will be noted pertaining this aspect during my final interaction with him/ her I will gladly fill in before his/ her departure from the hospital. Results and recommendations of this study will also help doctors understand heart failure patients better and audit themselves concerning the health education they are offering their (heart failure) patients so that they can improve on the same in the care of future patients.

Participation is voluntary and you are free to withdraw your child from the study at any time during the course of this period. Your refusals to let him/ her participate or withdrawal from the study will not in anyway affect the quality of his/her treatment.

All the information obtained will be treated confidentially.

I,.....parent/ guardian to .....
understand the above and voluntarily accept to allow my child participate in the study.
Signed.....Date.....

I confirm that I have explained to the parent/ guardian the above
Signed.....Date..... (Interviewer)

Telephone Contacts (of parent/guardian).....

APPENDIX 5 STUDY PROFOMA

STUDY No..... WARD..... ROOM.....

A. PATIENT DETAILS

i. Date.....

ii. Hospital no.....

iii. .Sex..... MALE = 1 FEMALE = 2

iv. Age.....

v. District of birth.....

vi. Residence within past 5 years.....

vii. Formal Education level (Tick one)

A. NONE =0

B LOWER PRIMARY = 1

C UPPER PRIMARY=2

D. SECONDARY=3

E COLLEGE/DIPLOMA=4

F GRADUATE =5

***B.* HISTORY**

(i) Symptoms	present (=1)	absent (=0)
A. Shortness of breath on exertion.....		.....
B. Easy fatiquability.....		.....
C. Palpitations.....		.....
D. Night cough.....		.....
E. Leg swelling.....		.....
F. Haemoptysis.....		.....
G. Orthopnea.....		.....
H. Night sweats.....		.....
I. Abdominal swelling.....		.....
J. Fever.....		.....
K. Weight loss.....		.....
L. Anorexia.....		.....
M. Yellowness of eyes.....		.....
N. Vomiting.....		.....

(ii) NYHA Functional class.....

(iii) Past history of admission for heart failure

YES=1	NO=0

(iv) Social/occupational history

A1 . History of past or current smoking (Tick one)

A2 If yes No. of pack years

YES=1	NO=0

<1/4	¼-1/2	1/2 – 3/4	¾-1
1- 5	6 -10	11- 20	>20

B1 History of past or current alcohol consumption

YES =1	NO =0

B2. If yes, what is the type of alcohol?

CHANG'AA	OTHER LOCAL BREWS	COMMERCIAL BEER	SPIRITS

B3 What is the unit of measure……..

B4 How many units per week ……

B5 For how many years……

C. Occupation/ employment status (Tick one)

STUDENT =1	EMPLOYED =2	RETIRED =3	NOT EMPLOYED =4

D. Marital status (Tick one)

SINGLE=0	MARRIED =1	SEPARATED/DIVORCED =2	WIDOWED =3

C. PHYSICAL FINDINGS AT RECRUITMENT

General examination at recruitment:

i. Systolic Blood Pressure.....

ii. Diastolic Blood Pressure.....

iii. Heart Rate.....

iv. Respiratory rate.....

v. Temp.....

vi. Weight...A... On admission.....Height.....

B...On day 5.....

C... Weight difference.....

D...On discharge.....

vii. Leg oedema ..... (+) .....(-)

viii. Jaundice..... (+) .....(-)

ix. Wasting..... (+) .....(-)

Cardiovascular system findings

i. JVP..... RAISED NOT RAISED

ii. Pulse: volume. HIGH NORMAL LOW

iii. Pulse rhythm .. REGULAR IRREGULAR

iv. Apex beat..... DISPLACED NORMAL DIFFUSE

v. Parasternal heave..... (+) .....(-)

vi. Murmurs..... (+) .....(-)

vii. Gallop..... (+) .....(-)

Abdominal findings

i. Ascites ... . (+) .....(-)

ii. Tender hepatomegally.... (+) .....(-)

iii. Hepatojugular reflux..... (+) .....(-)

Respiratory findings

i. Respiratory distress.... (+) .....(-)

ii. Basal rales ..... (+) .....(-)

iii. Pleural effusion ..... (+) .....(-)

Significant findings in other systems

.....

D. LABORATORY/RADIOLOGICAL FINDINGS

i. Haemogram

A. Hb.....g/dl

B. MCV.....fl

ii. Renal Function

A. Creatinine.....

B. Na+.....

C. K+.....

iii. Chest X-Ray findings

YES	NO

A_1 Gross cardiomegally

A_2 Cardiothoracic Ratio.....

B.Kerly B lines	YES =1	NO =0
C.Pleural effusion	YES =1	NO =0
D.Alveolar oedema	YES =1	NO =0
E . Cephalization	YES =1	NO =0

E. HEALTH EDUCATION/ EXIT INTERVIEW

Have you received information from the medical team while in the Ward concerning the following as pertains to your heart problem:

		Received (+ =1)	Not Received(- =0)
i.	Weight monitoring.....	.....	
ii.	Alcohol abstinence	.....	
iii.	Avoidance/ quit smoking.....	.....	
iv.	Regular follow up at out patient clinic.....	.....	
v.	Drug compliance.....	.....	
vi.	Role of exercise.....	.....	
vii.	Reduction of salt intake.....	.....	
viii.	Reduction of fluid intake	.....	

F. DISPOSITION

i. Discharged on (date).....

ii. Died on (date).....

APPENDIX 6

NORMAL REFERENCE VALUES

Clinical

Heart rate.....50 – 100 beats/min

Respiratory Rate.....14- 16 breaths/min

Systolic Blood pressure 90-120mmHg

Diastolic blood pressure 50- 90mmHg

Jugular venous pressure ..... <4cm above sternal angle

Laboratory

Haemoglobin.....12-16 g/dl (female)

.....13-18g/dl(males)

Creatinine.....67-120micromol/l

Potassium.....3.5- 5.5mmol/l

Sodium.....135-145mmol/l

MCV.....80-96fL

Radiolological/CXR

Cardiothoracic ratio(CTR).....< 0.5